nificantly less than the cumulative risk of strut fracture during their expected lifetime.

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Abnormal right heart filling after cardiac surgery

SIR,—Dr Wranne and colleagues demonstrated in figure 5 of their interesting study that the lateral aspects of the tricuspid annulus showed a more pronounced motion loss after cardiac surgery than those of the mitral annulus.[1] As one of the possible explanations they suggested that the left ventricle was better preserved during surgery than the right ventricle. This theory is confirmed by an experimental study of the tissue electrolyte content in the right and left ventricular myocardium after normothermic open heart surgery in dogs.[2] Cardiac arrest had been induced (*a*) by clamping the ascending aorta, (*b*) by aortic clamping with additional injection of a cardioplegic solution, (*c*) and by electrically induced fibrillation (with preservation of the coronary circulation). Tissue electrolyte content was determined before extracorporeal circulation was started, as well as after an hour of recovery from a cardiac arrest of 30 or 45 minutes. In all these forms of cardiac arrest, tissue water had increased and potassium and magnesium decreased. These changes were more pronounced in the myocardium of the right ventricle in all experimental groups. A decrease in potassium and magnesium content in tissue is an indicator of cellular injury.[3–5] In the study in dogs the loss of these electrolytes was more pronounced in the myocardium of dogs with low cardiac output than in animals with adequate circulation after cardiac arrest. Because the dogs did not have genuine cardiac surgery cardiac arrest was relatively short and hypothermy was not used. Hence we do not believe that the observed differences between the ventricles were predominantly caused by a mechanical impediment, more pronounced exposure of the right ventricle to room temperature, or heat radiated from the operating room lights, as suggested by Wranne *et al*. We attribute this phenomenon to a proposed difference in the susceptibility of the right and left ventricular myocardium to systemic disturbances, as it has been described for various diseases, such as hyperosmolar coma or liver failure.[3 4 6] Histologically, ultrastructurally, and biochemically, the right ventricular myocardium differs from that of the left ventricle.[4 6 7] According to Doerr the different susceptibility of the ventricles to disease can be explained by phylogenesis: the right ventricle belongs mainly to the priscomyocardium and is phylogenetically older than the left ventricular neomyocardium.[6] Perhaps this so-called "theory of pathoclisis" also explains the differences between the right and left ventricular function seen after cardiac surgery and described by Wranne *et al*.

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Working party report on cardiac rehabilitation

SIR,—I was most interested to read the report from the working party on coronary rehabilitation and would like to congratulate them on their obvious hard work.[1]

Because coronary rehabilitation has not been a high technology subject it has lapsed into a cinderella type of service with a few enthusiastic doctors and many more enthusiastic nurses, physiotherapists, and occupational therapists soldiering on in isolation. It is for this reason that research has not been very forthcoming because individual units find that they do not have enough patients to produce meaningful controlled trials: for example, the trial by a Glasgow team in 1991 in the end had 12 patients in the treatment group and 10 in the control.[2]

Up to now it is the paper from O'Connor *et al* in 1989, which provided an overview of 22 small trials, that has allowed us to believe that coronary rehabilitation had a part to play in the treatment of acute myocardial infarction.[3]

Your readers may be interested to know that here in the North-West we have had the Coronary Rehabilitation Development Organisation (CReDO) for about two years now. It is a loose knit confederation of just such enthusiasts as I have described who meet quarterly to exchange ideas and support others who are in the process of setting up a rehabilitation programme.

Three things have become apparent. Firstly it requires a dedicated nurse/physiotherapist/occupational therapist around whom the whole programme pivots; secondly, it does not need much money to set up a simple scheme; and thirdly, and probably the most important, any scheme to be successful needs the wholehearted support of the consultant in charge of cardiology for that district—whether cardiologist or general physician with an interest in cardiology.

A meeting is to be held on 26 September 1992 in Oxford to try to organise a national association that may well then allow us to produce some proper scientific evidence to persuade those who control the budgets that rehabilitation is a worthwhile and cost-effective part of the treatment of myocardial infarction.

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BOOK REVIEWS

Heart disease: A textbook of cardiovascular medicine. 4th ed. Edited by Eugene Braunwald. (Pp 1874; $119.) Philadelphia, London, Toronto, Montreal, Sydney, Tokyo: W B Saunders Company, 1992. ISBN 0-7216-3097-9.

When the original edition of this comprehensive textbook appeared, I was given the opportunity to review it in 1980. It was the first of a new generation of such works, and set the standards by which others might be judged. Not only did other important similar works respond by improving their quality but a number of other textbooks have appeared, mainly in the United States though with one British based offering.

As with previous editions, Braunwald is an author or co-author of many of the chapters as well as having orchestrated the whole. He has taken considerable trouble to ensure that what appears is up to date even at the cost of shedding some earlier references from some chapters that may be of historical relevance. But as his purpose is to present contemporary evidence, this has enabled him to limit the size to some extent. Nevertheless, there are still 1874 pages of text and illustrations, with ample and appropriate references.

The whole of the subject is covered extensively and if some miss their favourite topics, that will be highly exceptional. For a description of pathological processes generally one will have to turn elsewhere and this is probably appropriate in a book aimed at providing clinically relevant information, yet readers should not forget the potential importance of such knowledge.

There have been modest changes in authorship once again between the third and fourth editions, and Braunwald's system of having all the chapters externally reviewed ensures a high standard throughout.

The most striking feature of the present edition is the lavish use of colour. Generally this is helpful and indeed to have the chapter numbers coloured red discriminates them from the pages above and makes for the easier finding of what one needs from an index that did not fail several random tests. Those interested in electrocardiography will, however, question the use of red for the tracings and a dark but absolutely crisp background for the grids (the latter is an excellent feature); surely it would have been easier for most if the conventions had been observed and the tracings had been dark and the background coloured as you see in clinical tracings. In other respects I accept, and on the whole welcome, the use of colour, which